

LETTER TO THE EDITOR

Chemical gastritis and colitis related to
hydrogen peroxide mouthwash

Correspondence Moira Ragazzi, MD, Anatomic Pathology Unit, Arcispedale Santa Maria Nuova – IRCCS, Viale Risorgimento, 80, 42123 Reggio Emilia, Italy. Tel.: +39 05 2229 5657; Fax: +39 05 2229 6945; E-mail: moira.ragazzi@asmn.re.it

Received 16 May 2016; **Revised** 11 August 2016; **Accepted** 19 August 2016

Magda Zanelli, Moira Ragazzi and Loredana De Marco

Pathology Unit, Arcispedale Santa Maria Nuova – IRCCS, Reggio Emilia, Italy

Tables of Links

TARGETS
Voltage-gated ion channels
TRPM2

LIGANDS
Hydrogen peroxide

These Tables lists key protein targets and ligands in this article that are hyperlinked to corresponding entries in <http://www.guidetopharmacology.org>, the common portal for data from the IUPHAR/BPS Guide to PHARMACOLOGY [1], and are permanently archived in the Concise Guide to PHARMACOLOGY 2015/16 [2].

Hydrogen peroxide is a commonly used oxidizing agent with different uses depending on its concentration. Three percent solutions are used as common household disinfectants for superficial wounds. Hydrogen peroxide has been utilized widely throughout medical history. Clinical applications involving the gastrointestinal tract have included rectal enema to relieve faecal impactions in adults or meconium ileus in newborns. Nowadays most of these practices have been abandoned due to severe intestinal iatrogenic injuries [3]. Ingestion of 3% hydrogen peroxide is usually considered to be relatively nontoxic, producing only minimal gastrointestinal irritation [4].

We report a case of chemical gastritis and colitis after use of mouthwash with 3% hydrogen peroxide solution. Patient consent was obtained.

A 54-year-old woman presented to our institution complaining of acute epigastric and abdominal pain associated with postprandial, nonbloody diarrhoea. She also suffered from mild pharyngalgia and a sore mouth. The symptoms had lasted for about 2 months becoming progressively worse. The patient was afebrile. Her past medical history was unremarkable. She denied taking any medication. Physical examination revealed a mild diffuse abdominal tenderness. Her blood examinations were within normal limits. Upper and lower gastrointestinal endoscopy did not reveal any abnormalities. Multiple gastric biopsies showed mild gastritis with focal superficial erosions.

Microscopic examination of multiple colorectal biopsies revealed mucosal oedema, vascular congestion and mild gland atrophy. An increase in apoptotic bodies in the cryptal epithelium of the colorectal mucosa was also noted. The pathological features appeared consistent with mild ischemic colorectal damage. However, the presence of apoptosis in the crypt epithelium raised the possibility of a drug effect [5]. A detailed clinical history disclosed that the patient was having an endodontic root canal procedure to treat an infected tooth when the gastrointestinal symptoms had started. The endodontic treatment was performed once a week for 2 months. During each session 50 ml of a 3% hydrogen peroxide solution was applied to clean out and disinfect the root canal. Isolation devices to prevent the patient from swallowing the product, such as dental dams, were not used and presumably a large amount of the hydrogen peroxide solution was ingested. The gastrointestinal symptoms had started approximately 4 weeks after the beginning of the endodontic procedure and had become progressively worse during the 2-month treatment period. After suspension of the mouthwash the patient achieved a complete resolution of gastrointestinal symptoms.

The history of hydrogen peroxide toxicity in the gastrointestinal tract dates back to its use with enema causing severe colonic damage similar to that of ulcerative colitis or pseudomembranous colitis [6]. Cases of chemical colitis have also been reported to occur as a result of accidental

contamination of endoscopes by disinfecting solutions containing hydrogen peroxide [7]. Corrosive damage, oxygen gas formation and lipid peroxidation make up the major mechanisms of hydrogen peroxide toxicity.

Nowadays, hydrogen peroxide toxicity usually occurs secondary to accidental exposure. The concentration of hydrogen peroxide is considered an important risk factor for its toxicity [8].

Although ingestion of 3% hydrogen peroxide is normally nontoxic or produces only minimal gastrointestinal irritation, it should not be considered entirely innocuous [4]. The severity and extent of damage presumably depends on the volume ingested [4, 9]. Most exposures to 3% hydrogen peroxide occur in children, involve accidental ingestion of <30 ml and cause only minor symptoms. However, rare cases of gastrointestinal bleeding and gas embolism have been described in children following ingestion usually of greater volumes of 3% hydrogen peroxide [4, 9]. To the best of the authors' knowledge this is the first report documenting chemical gastritis and intestinal ischemic damage, following ingestion of a diluted hydrogen peroxide 3% solution repeatedly used as an antiseptic during an endodontic treatment. The damage is presumably related to both the volume swallowed and the chronic nature of use. The proposed mechanism of toxicity could be direct corrosive damage in the stomach and an accumulation of free radicals in the intestine, generated by hydrogen peroxide catabolism. Free radicals induce both membrane injury through lipid peroxidation and vascular smooth muscle contraction influencing mucosal blood flow [10]. Hydrogen peroxide is commonly used at low concentration as an antiseptic mouthwash and tooth-bleaching agent [11]. Therefore, it is important for clinicians to be aware of the potential damage that can be caused to the gastrointestinal mucosa if hydrogen peroxide is inadvertently and repeatedly swallowed, as it was in our case. Given the nonspecific symptoms of chemical induced gastritis and colitis, the diagnosis can be challenging if the pertinent history is not obtained.

Competing Interests

All authors have completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf and declare: no

support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous 3 years; no other relationships or activities that could appear to have influenced the submitted work.

References

- 1 Southan C, Sharman JL, Benson HE, Faccenda E, Pawson AJ, Alexander SP, *et al*. The IUPHAR/BPS Guide to PHARMACOLOGY in 2016: towards curated quantitative interactions between 1300 protein targets and 6000 ligands. *Nucl Acids Res* 2016; 44: D1054–D1068.
- 2 Alexander SPH, Catterall WA, Kelly E, Marrion N, Peters JA, Benson HE, *et al*. The Concise Guide to PHARMACOLOGY 2015/16: Voltage-gated ion channels. *Br J Pharmacol* 2015; 172: 5904–41.
- 3 Sheibani S, Gerson LB. Chemical Colitis. *J Clin Gastroenterol* 2008; 42: 115–21.
- 4 Henry MC, Wheeler J, Mofenson HC, Caraccio TR, Marsh M, Comer GM, *et al*. Hydrogen peroxide 3% exposures. *J Toxicol Clin Toxicol* 1996; 34: 323–7.
- 5 Lee FD. Importance of apoptosis in the histopathology of drug related lesions in the large intestine. *J Clin Pathol* 1993; 46: 118–22.
- 6 Love BL, Siddiqui S, McCallum BJ, Helman RM. Severe chemical colitis due to hydrogen peroxide enema. *J Clin Gastroenterol* 2012; 46: 87.
- 7 Jonas G, Mahoney A, Murray J, Gertler S. Chemical colitis due to endoscope cleaning solutions: a mimic of pseudomembranous colitis. *Gastroenterology* 1988; 95: 1403–8.
- 8 Dickson KF, Caravati EM. Hydrogen peroxide exposure – 325 exposures reported to a regional poison control center. *J Toxicol Clin Toxicol* 1994; 32: 705–14.
- 9 Rackoff WR, Merton DF. Gas embolism after ingestion of hydrogen peroxide. *Pediatrics* 1990; 85: 593–4.
- 10 Geboes K, De Hertogh G, Ectors N. Drug-induced pathology in the large intestine. *Curr Diagn Pathol* 2006; 12: 239–47.
- 11 Walsh LJ. Safety issues relating to the use of hydrogen peroxide in dentistry. *Aust Dent J* 2000; 45: 257–69.